Sorghum Diseases in West Africa

An Illustrated Text

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Foreword

The Regional Food Crop Protection Project (RFCP) is a United States Agency for International Development (USAID) funded project in West Africa. Participating countries have included Cameroon, Cape Verde, Chad, the Gambia, Guinea-Bissau, Mali, Mauritania, and Senegal.

Starting in 1976, the project's objective has been to improve the capability of the participating countries to deal with a variety of plant pest problems affecting food crops both in the field and in storage. These pest problems have included weeds, insects, plant diseases, nematodes, and vertebrate pests such as rodents and birds.

Improving the national capability to deal with food crop pest problems has required RFCP to carry out activities with national plant protection services, national agricultural research institutes, extension services, and various agricultural educational institutions. These activities centered on what the farmer could do for himself, what government could do that was beyond the capability of the farmer, and emergency pest outbreaks.

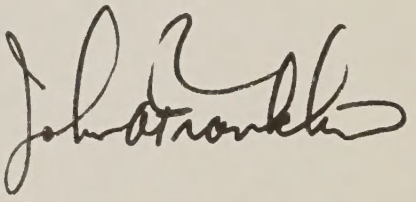
One area of RFCP activity centered on identifying and publishing informational and training materials. When the project began in 1976, it was obvious that very few materials existed that could be drawn on by government workers as they attempted to identify pest problems and recommend appropriate controls. To help meet this need the project has sponsored this publication: *Sorghum Diseases in West Africa.* It is our hope that it will also be published in French so its potential audience of government workers can be expanded.

While RFCP has been staffed by employees of the U.S. Department of Agriculture (USDA), Animal And Plant Health Inspection Service (APHIS), Plant Protection and. Quarantine (PPQ) division, credit must be given also to USDA's Agricultural Research Service and Office of International Cooperation and Development; numerous universities, particularly Oklahoma State University; and USAID, Office of Agriculture, Science and Technology, and the many USAID employees who have been so helpful over the years.

Acknowledgment must also be made of the cooperative efforts of international agricultural institutions such as the International Institute for Tropical Agriculture in Nigeria.

A special thanks goes to USDA-APHIS-Information division for their assistance in producing this publication for the project. Without them this text would not exist.

Suggestions or inquiries regarding this publication should be addressed to RFCP, care of USAID, BP 49, Dakar, Senegal.



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Western Africa, from the aspect of sorghum diseases, can be considered a single integral unit. There are no physical barriers to the movement of sorghum diseases throughout this area. The harmattan (a dry, dusty wind that blows from the Sahara and along the northwestern coast of Africa) and the winds that precede the early season rains have no respect for national frontiers. They can pick up plant pathogens on plant debris in the soil and carry them considerable distances. In addition, the movement of sorghum seeds over the commercial trade routes through West Africa in past centuries has disseminated most of the indigenous plant pathogens throughout the region, so that they are restricted in their occurrence only by the local effects of environment.

This text has been prepared to aid those who wish to identify or know more about sorghum diseases in West Africa. It was written for plant pathologists, plant breeders, agronomists, and extension and Ministry of Agriculture personnel who have a need to identify sorghum diseases in the field. The disease descriptions and photographs are, therefore, designed for this purpose. For laboratory identifications of the pathogens that incite these diseases, the reader is advised to consult the excellent works of Saccas (*25*) and Tarr *(27).*

Sorghum diseases in West Africa in the main are similar to sorghum diseases in the United States *(7, 16),* although some unique disease situations occur in West Africa. Although in this illustrated text the sorghum diseases in Nigeria have naturally been stressed, available information on sorghum diseases in other countries of West Africa has also been included.

The author expresses his appreciation to the many who have been of assistance, not only in preparing this text, but also in carrying out research at Ahmadu Bello University, Zaria, Nigeria. These include: Dr. L. K. Edmunds, Dr. R.A. Fredericksen, Dr. M. ElRouby. Dr. K. Starks, Mr. J. C. Girard, Mallam Imranah Yazidu, and Alhaji Dodo Mustapha. My special thanks go to Dr. D. McDonald, Professor of Crop Protection at Ahmadu Bello University, for his critical review of the manuscript. The author also has the pleasure of expressing his sincere gratitude to Mr. S.K. Manzo, his Nigerian counterpart, whose cheerful assistance and timely advice helped make this publication possible.

Appreciation is especially due to the Institute for Agricultural Research at the Ahmadu Bello University, which has been most generous in making available facilities necessary for the completion of this work.

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Photo Credits

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R. A. Frederiksen, Department of Plant Science, Texas A&M University, College Station, Texas (Figure 42).

Sorghum, great millet, or Guinea corn *(Sorghum bicolor* (L) Moench) is probably the most important food crop in West Africa. Along with pearl millet (*Pennisetum americanium* (L) Leeke), sorghum provides the main energy source for the people of the Guinea and Sudan zones *(2).**

Sorghum is well adapted to the climatic conditions in West Africa. Sorghum and millet can withstand periods of drought better than other major cereals, and they can resume growth when the rains come again. The crop fits in well with the 4- to 5-month rainy season and 7- to 8-month dry season of most of West Africa. The bulk of the sorghum crop is planted soon after the beginning of the rains in May and June and is harvested after the rains have finished in October and November. The dry season assists the sorghum crop by bringing about, through desiccation, the destruction of many diseases and pests, so that in theory the new crop starts off clean every year.

Dry-season sorghum is cultivated in flood plains, especially in Mali *(30)* and in Chad, Cameroon, Niger, and Nigeria around Lake Chad. The crop is transplanted in October from nursery beds sown during the rains in August. Harvest takes place in February and March.

A third type of sorghum culture is practiced in Mali *(30)* where the annual rainfall is less than 425 mm. The crop is sown directly in the fields in January or transplanted later from nursery beds. It becomes established before the rains but does not mature until they have finished in October. At present, most sorghum is intercropped, usually with millet; however, maize, cowpeas, groundnuts, cassava, and several other crops may also be included in the intercropping mixture.

In addition to the sorghum grown for grain for human consumption, several other forms of sorghum are cultivated or exist in the wild state. Tarkanda or sweet sorghum *(Sorghum bicolor)* with sweet, juicy stalks is grown for chewing, as with sugarcane.

*Italicized numbers in parentheses refer to the References at the end of the text.

Figure 1. Large head of white-seeded sorghum with grain matured after the rains ended.

Figure 2. Large head of yellow-seeded sorghum which is preferred in some areas of West Africa.

Sudangrass *(S. sudanense* (Piper) Stapf is grown for livestock feed (mainly horses). Johnsongrass *(S. halepense* (L.) Pers. grows wild in the more humid areas and is used as fodder. Broomcorn *(S. bicolor* (L.) Moench produces long, brush-like heads and is cultivated for the manufacture of brooms and brushes.

The origin and classification of sorghum is discussed by Garber *(12),* Snowden *(26),* and De Wet and Harlan *(4).* Local sorghum varieties belong to the Guinea and Kafir races. However, plant introductions from the United States and other parts of the world have brought in other races *(9).*

Most local sorghum varieties in West Africa are photosensitive and respond to changes in day length. Also, their heading date coincides with the end of the rainy season, which avoids head mold and weathering of the grain. Therefore, their growing period gets longer as one moves toward the equator. Because of this, the varieties are narrowly adapted and there is a gradual shift of varieties over an area of about 50 miles north or south. Also the preference for seed color and grain type varies from one location to another. All of these factors result in a wide range of variability.

There is a considerable variation in the type of sorghum grown in West Africa. Most sorghum varieties are tall stemmed, late maturing (4–8 months), usually with loose, open panicles with large seeds. The white-seeded varieties (fig. 1) are preferred in most areas, but yellow- (fig. 2) and tan- or brown-seeded varieties are grown in some areas. Red-seeded varieties are preferred for beer making. The local varieties are low yielding, adapted to low fertility, and generally not responsive to high levels of fertilizer. They are grown under low plant population (10,000 per acre) to avoid exposing the crop to moisture stress.

Importance of Sorghum Diseases

It is an irony of agriculture that as cultivation of a given crop species becomes intensified, its vulnerability to pests and diseases also increases.

Diseases of sorghums, like those of other crops, vary in severity from year to year and from one locality or field to another depending on environment, causal organisms, and host resistance. Misconceptions of the nature of disease often arise because the interdependency of these factors is not adequately considered in the study of disease development.

Environment is critical to disease development to a degree that varies considerably among individual diseases. In general, soil temperature and moisture are most critical for soilborne pathogens, which attack roots and stalks or infect young shoots before they emerge. Air temperature, moisture, wind, and light are most critical for airborne pathogens, which attack foliage, stalks, and inflorescences.

No disease will occur, however, if the causal organisms or pathogens—virus, bacterium, fungus, or nematode—are not present, even though the environment is favorable for disease development and the host is susceptible. The causal organisms may vary in virulence as much as the hosts may vary in relative resistance. Some pathogens, especially those attacking roots and stalks, may have little or no specificity, and may attack many plant species in addition to sorghum. Others, such as smut fungi, are so highly specific that some forms can attack only certain varieties of sorghum. Most pathogens, however, have intermediate specificity: they may be able to attack any of several crops; they may only attack maize, millet, sorghum, and related grasses; or they may be limited to attacking only certain types of sorghums. As the intimacy between parasite and host increases, so usually does the specificity of both for each other.

Resistance or susceptibility of various sorghum genotypes to a given pathogen often reflects the genetic nature of virulence in the pathogen. For example, resistance to the common race of the head smut fungus *(Sphacelotheca reiliana*) is imparted by a single dominant gene *(24).*However, this single gene resistance can be overcome simply by the buildup of a race of the pathogen that can attack varieties with this resistance. Such genetic resistance is used in cultivation when the pathogen has little capability for producing new strains or when quick (although temporary) control is needed *(29).* For such a disease as charcoal rot *(Macrophomina phaseoli)*, on the other hand, such single-gene resistance is not practical because the pathogen involved has little specificity for the plant species or varieties it attacks. Developing resistance to such a pathogen is difficult because there is little chance of finding resistance that is simply inherited. If, however, it is possible to isolate genotypes that are tolerant to such a nonspecific pathogen, such resistance probably would be durable and would justify the extra time and effort required to obtain it.

Coexistence with Sorghum Diseases

Sorghum probably always will have diseases. It is not feasible to eradicate them, but it is possible to minimize disease losses by discrete control methods requiring cooperation of those most directly concerned with sorghum production—farmers and extension and research personnel.

Farmers are in the best position to monitor crop health and report any decline or abnormality, as soon as observed, to extension workers or experiment stations. The relative importance of disease problems could be evaluated much better by the plant pathologists and breeders responsible for developing control measures if they were aware of such observations.

Control of sorghum diseases depends heavily on development of disease-resistant genotypes, using simple and multigenic resistance available in many breeding nurseries and in the world collection of sorghum germplasm *(22).* Usually, resistant lines first are identified in nurseries established at locations where each disease can develop naturally. When incidence of a given disease is high among susceptible genotypes in such nurseries, it is relatively simple to identify and preserve the resistant genotypes. If, however, using natural infection to identify resistance is either too slow or too unreliable, it may be necessary to inoculate artificially and produce epidemics under controlled conditions. Artificial inoculation is particularly effective if reactions to a particular disease can be obtained from seedlings to minimize time and space requirements.

Incorporating identified resistance is a final step in developing resistant hybrids or varieties. When a single disease is involved, resistance, controlled by one or a few genes, can be incorporated by simple crossing and backcrossing. When resistance to several diseases or multiple-gene resistance to few diseases are incorporated into one genotype, other methods must be used. Currently, the recurrent selection is suggested and is feasible in

sorghum by use of suitable male steriles *(9)*. Resistance inherent in many selections can be brought together in a single genotype.

Cultural control methods may sometimes be used when resistance to disease is unavailable or incomplete. New strains of highly specific pathogens that can reproduce rapidly or to which sorghum cultivars have no specific resistance, may require this type of control *(29)*. For example, one should avoid growing (and incorporating into the soil) varieties super-susceptible to such diseases as downy mildew *(Peronosclerospora sorghi)* and head smut, which are initiated each season by soilborne spores. Destroying diseased plant refuse may reduce losses from some foliage pathogens if fields are isolated sufficiently. Little is known of the effect of some cultural practices on the increased incidence of those diseases may relate to heavy applications of nitrogen or the use of some acid-forming fertilizers.

Sometimes weeds such as volunteer crop plants or perennial wild grasses may serve as carryover hosts in perpetuating diseases from one cropping season to another. There is an ethic in West Africa that no food plant be destroyed. This may have to be modified somewhat especially as more irrigation schemes come into being. Diseased volunteer crop plants and weeds that are permitted to come through the dry season in such irrigation schemes will serve as inoculation centers for disease when the rains come.

Chemical control of disease in sorghum in West Africa is not feasible, except by seed treatment for such diseases as the kernel smuts, seed-rot, damping-off, and seedling blight. Development of new systemic fungicides with long-lasting protection, however, may lead to better means of chemical control in the future.

Young sorghum plants are rather delicate and grow slowly. It takes them at least 10 days to establish permanent, adventitious roots *(10)*. Until then, anchorage and nutrients must be obtained by a relatively diminutive and transitory primary root system and a few adventitious roots arising from the coleoptile nodes. Under less than optimum growing conditions, that root system is highly vulnerable to soilborne pathogens.

Sorghum mortality during the first month after planting is a problem in many areas. Laboratory germination tests do not indicate field germinability as effectively with sorghum seed as with other cereal seed. Fifty to seventy percent germination is the best that can be expected in the field. Sorghum seed planted under unfavorable conditions for germination and subsequent seedling growth quickly rot in the soil and must be replanted. Although soil crusting or surface desiccation sometimes can be blamed, seedborne fungi that attack seed or seedling parts usually are responsible. Cold rains, lack of sunshine, poor drainage, poor soil aeration, competition with weeds, and toxic plant residues can offset seed treatment given as protection against those fungi. Insects and birds also play an important part in reducing seedling emergence. Failure to get good stands often leads to excessively high seedling rates, which in turn can introduce other problems such as seed rot, seedling diseases, and stress-related diseases at later stages of growth. *Fusarium moniliforme* and *Phoma sorghina* can cause severe reductions in stands of young sorghum plants, especially during periods of wet, cloudy weather where there is little sunshine for several days. These fungi may cause a seed rot which reduces germination or they may attack and kill seedlings after the young plants emerge. Seedling leaves become spotted and brown; and the plants die before they become established. Some soil fungi such as *Sclerotium rolfsii* and *Aspergillus niger*, especially when carried on the seed, can completely inhibit germination *(17)*.

Because sorghum seeds are closely grouped in large panicles and large portions of the seed are directly exposed to the environment, they provide an ideal site for fungal growth, especially if humid conditions prevail as the grain matures. Most local sorghum varieties such as 'Fara Fara' and 'Short Kaura' develop their grain after the rains end in normal years. This enables them to produce grain that is virtually free from seed molds. However, during the drought period in the Sahel from 1969 to 1973, some of these long-season, photosensitive varieties failed to produce satisfactory crop yields because the rains ended too soon. This led to the introduction of short-season, non-photosensitive varieties that mature grain before the end of the rainy season, which in turn complicated the seed mold problem (fig. 3). Grain mold damage reduces grain quality and such grain is especially undesir-

Figure 3. Seed mold on sorghum head.

able for human consumption. If early-maturing sorghums are to succeed as a source of human food in West Africa, it is essential that they be resistant to grain molds. Some of the introduced short-season sorghum varieties from the World Collection in India and Nigeria possess a high degree of seed and mold resistance *(35)*. Potentially, these can be incorporated into shortseason, high-yielding varieties adapted to West Africa. Date of planting of short-season varieties plays an important part in the amount of seed mold infection. Early plantings with more exposure of the maturing seed to the rains will normally have more seed mold present than will later plantings. However, sorghum varieties that are superior in grain mold resistance will remain relatively resistant to grain molds whether planted early or late. The exact nature of grain mold resistance in sorghum is still not clearly defined. Sorghum varieties with a hard, corneous, or waxy seed coat tend to show more seed mold resistance than those with a soft, chalky, or mealy-type seed coat. Varieties with loose, open heads, to a degree, show more seed mold resistance than do varieties with dense, compact heads. Some of the red-colored, high-tannin varieties are less prone to attack by certain seed fungi in the field than are some white-seeded, low-tannin varieties.

Manzo *(18)* at Samaru, Nigeria, showed an association of several seedborne fungi with seed of certain sorghum varieties planted in the same field in 1975. When fungi isolated from three varieties were compared, *Phoma sorghina* was isolated from 36 percent of 'Short Kaura' seed, 37 percent of 'Samaru 2123' seed, and 90 percent of 'Roma' seed. *Fusarium moniliforme* was isolated from 2 percent of 'Roma' seed, 4 percent of 'Samaru 2123' seed, and 37 percent of 'Short Kaura' seed. Manzo *(18)*. found that the most commonly isolated fungi from sorghum seed in Nigeria, in relative order of frequency, were *Phoma sorghina, Fusarium moniliforme, Curvularia lunata, Helminthosporium halodes, Aspergillus niger, Chaetomium sp., Penicillium funiculosum Cladosporium sp., Rhizopus stolonifer, Fusarium semitectum, Collectotrichum graminicola, Aspergillus flavus, Nigrospora sphaerica, Alternaria alternata, Sclerotium rolfsii, Fusarium oxysporum*, and *Fusarium culmorum*. The action and interaction of those fungi on seed in storage is not fully known. It can be assumed, however, that under conditions of relatively high humidity, they can cause seed rot, decrease the food quality of the seed, and reduce germination.

Root Rot

Sorghum root rot is commonly found in all sorghum-growing areas. The extent of damage to the crop is greatly influenced by soil and environmental factors. Root rot is decidely more important on infertile, poorly drained, or very acid soils than on fertile, friable, well-drained soils.

The importance of sorghum root rot in West Africa is generally underestimated, because the damage is below the soil surface and is hidden from the eye of the casual observer. Poor plant growth in many cases of root rot is commonly explained as being due to poor soil fertility, especially when such plants do not show any serious above-ground disease symptoms. When a sorghum plant affected with root rot is removed from the soil, the roots, particularly the young, fine ones, are rotted or dead (fig. 4). Early symptoms of root rot appear as small, circular to oblong, red-brown to purple-black lesions on the root surface *(19)*. The lesions become larger and deeper until the conductive portion of the root is severed by the lesion. The cortex, or outer layer, of diseased roots easily separates from the stele, or central conductive portion, of the root. All sorghum varieties will show some fungal lesions and rotting of the cortex and still be able to produce a normal yield. If, however, the root rot extends into the stele, the conductive ability of the root is impaired and the plant suffers. Sorghum varieties in which root lesions are confined to the cortex may generally be considered resistant to root rot, whereas varieties in which the rot localizes in the stele should be considered susceptible.

Numerous fungi in the soil are capable of causing root rot in sorghum. Manzo *(18)* in Nigeria found that *Fusarium oxysporum, Trichoderma harzianum, Fusarium moniliforme, Curvularia lunata, Macrophomonia phaseoli, Penicillium*

Figure 4. Sorghum root rot on a root-rot susceptible variety.

funiculosum Rhizopus stolonifer, Aspergillus niger, Helminthosporium halodes. Sclerotium rolfsii, Verticillium sp. Aspergillus flavus, Fusarium culmorum, Rhizoctonia solani, Fusarium solani, Cunninghamella echinulata, Phoma sorghina, Helminthosporium rostratum, Cephalosporium sp., Periconia macrospinosa, and *Nigrospora sp.* were able to cause root rot in sorghum. In addition he found at least two bacteria that were also associated with root-rotted plants. The extent of damage from root rot depends on the interaction of the ability of the pathogenic fungi in the soil to attack sorghum roots and the ability of the sorghum plant to produce new roots without having its growth retarded. The fine young feeder roots, which are the main sources of water and mineral absorption in the plant, are generally the first attacked and destroyed. If a sorghum plant is vigorously growing in good soil under favorable environmental conditions the plant is able to withstand the loss of some feeder roots. Such a plant quickly responds to such root loss by producing new ones to replace the killed or injured ones. If, however, the plant is exposed to poor growing conditions or conditions of stress, it remains weakened and will be unable to produce new roots to replace the rotted ones. Eventually, if enough roots are killed and not replaced, the overall growth of the plant wil be severely retarded. It may die from inability to absorb enough water and nutrients from the soil.

Control of sorghum root rot consists of reducing damage to the plants by modifying factors that influence root rot. Some of the Nigerian local varieties such as 'Fara Fara', when properly spaced, are more tolerant to root rot than are some of the exotic varieties planted under high plant populations. Proper surface drainage to eliminate water logging of the soil and control of excess acidity or alkalinity of the soil will also help in reducing root rot.

Anthracnose, Stalk Red Rot, and Peduncle Breakage

Anthracnose, stalk red rot, and peduncle breakage are phases of a disease incited by *Colletotrichum graminicola* (Cesati) G. W. Wilson (15). All phases may occur at one time or only one or two may be present. This disease, which also attacks maize and several other grasses, is generally most prevalent in areas where daily periods of high humidity alternate with relatively dry periods. Normally, the disease is not found in very humid, very dry, or irrigated areas. The anthracnose phase, which may occur at any stage of plant development, usually occurs when the plants begin to form joints. It appears on the leaves of susceptible varieties as small, sunken, circular-to-elliptical spots 3 to 6 mm in diameter (fig. 5). The

Figure 5. Foliar anthracnose on sorghum leaves.

Figure 6. Anthracnose on midribs of sorghum leaves.

Figure 7. Red rot of sorghum stalks. Note the discrete white areas which are characteristic of the disease.

spots are well defined and, depending on the variety affected, may be tan, orange, red, or blackish-purple. Under conditions of high humidity, the spots increase in number and enlarge to cover much of the leaf area. Midrib infection commonly occurs as elliptical to elongate, discolored lesions, which frequently coalesce to cover the entire length of the midrib (fig. 6). On many of those spots, a blackish growth (visible to the naked eye) is produced. When examined under magnification this growth is seen to be made up of many short, stiff hairs (setae) that are part of the fruiting bodies of the fungus. Anthracnose kills leaf tissue and may defoliate sorghum plants completely, thus reducing growth and further development.

The very aggressive development of this disease, frequently leading to total leaf destruction, usually begins at the jointing stage. Typically, with the initial infection occurring on the older leaves in August in northern Nigeria, the disease progresses rapidly on very susceptible varieties, spreading up the plant so that the foliage may be completely destroyed by flowering time. On less susceptible varieties, the time of total leaf destruction may be delayed until after the seed has matured. Midrib infection can also occur at any stage of plant growth, although as with foliar anthracnose, the more aggressive development takes place on older leaves. All degrees of reaction to anthracnose occur among the sorghum varieties grown in West Africa and range from very susceptible, characterized by complete leaf destruction at or before flowering, to almost immune, in which no leaf spotting develops.

The red rot phase primarily affects stalks of mature sorghum plants. Spores of the fungus produced on the leaves in the anthracnose phase are washed downward behind leaf sheaths by rain. These spores germinate and the fungus enters the stalk at any time after the jointing stage and rots the interior of the stalk. Splitting diseased stalks lengthwise reveals discolored areas interspersed with white, which gives a marbled appearance throughout the affected portions (fig. 7). Stalk lesions are prevalent on the peduncle and internodes of the upper half of the plant. Depending on the variety affected, the discolored areas range from tan to purplish red. Diseased stalks frequently break near the middle of the stalk or just below the seed head. Diseased, but unbroken, stalks often produce small heads, sometimes with abnormally small seeds. Badly rotted stalks of some susceptible varieties sometimes result in total loss of the crop.

Figure 8. Peduncle lesions incited by *Colletotrichum graminicola* on sorghum.

Peduncle breakage sometimes occurs on susceptible sorghum varieties without the presence of leaf anthracnose or red rot of the stalk. The exact nature of the method of infection is not clearly understood, but apparently the fungus can enter the peduncle by direct penetration of the rind. Isolated lenticular-shaped lesions, ranging from 4 to 6 mm in diameter to the entire thickness of the peduncle, develop just beneath the epidermal tissue (fig. 8). The fungus spreads to the interior of the rachis and rachilla branches, interfering with movement of water and food materials, thereby causing poor development of heads and grain. The dry, rotted tissue shrinks and the peduncle breaks at the lesion.

The fungus survives from growing season to growing season in the soil, on plant refuse, or in susceptible weeds. In addition, the fungus may be carried on the seed and may attack young seedlings. Fortunately, sorghum varieties resistant to all phases of the disease are available (fig. 9). All newer sorghum varieties are highly resistant to anthracnose, red rot, and peduncle breakage. The broomcorns as a rule are susceptible, but they are grown in areas where the disease generally is not prevalent, thereby minimizing losses. Using clean seed, destroying plant refuse from previous susceptible crops, and controlling susceptible weeds also help to control the disease.

Bacterial Soft Rot

Bacterial soft rot incited by an *Erwinia* sp. (probably *Erwinia dissolvens* [Rosen] Burkholder) is found sporadically in West Africa *(31)*. Diseased plants in the field appear moribund; upper whorl leaves may be dead, but some of the lower leaves may appear unaffected. In some aspects, early symptoms of bacterial soft rot resemble shoot fly injury, in that the tops of affected plants are quickly killed. Diseased stalks are soft to the touch and break readily when handled. A strong putrid odor is associated with rotted stalks. When stalks that are not completely rotted are split lengthwise, hollow areas (gas pockets) 25 to 125 mm long by 12 to 18 mm in diameter are found throughout, some at the nodes and others

Figure 9. Anthracnose on a plant from a susceptible sorghum variety compared with no reaction on a plant from a highly resistant line.

between the nodes (fig. 10). A red ring of vascular bundles is found just inside the rind next to the hollow areas. Severely infected stalks, when rubbed between the fingers, are reduced to a rind and a mass of shredded, easily disjointed fibers. These are the remnants of the fibro-vascular systems.

The disease may attack plants at any stage of growth, but it is most noticeable on plants that have begun to form joints (3 to 5 feet tall). The disease kills quickly once infection starts; normally within 3 to 5 days after the first symptoms appear. Sometimes only the tops of plants are killed and shoots are produced from buds near the soil line. Bacterial soft rot also attacks maize in the United States.

Fortunately, although bacterial soft rot may be very severe on individual sorghum plants, only very few plants of susceptible varieties are infected. Most sorghum varieties appear to be highly resistant to bacterial soft rot. Control measures are not warranted.

Charcoal rot

Charcoal rot, incited by *Sclerotium bataticola* Taub., is more serious in drought years than in normal wet years. It can occur wherever the crop is subjected to low soil moisture and high temperature during seed development, because the fungus is present wherever sorghum is grown. The fungus attacks a wide variety of plants including the cereals, bananas, groundnuts, and tomatoes. The disease is more widespread in some years and localities than in others. The first plants affected generally are those in the drier soils where drainage is best, such as on terrace crowns, knolls, or soils underlain by coarse sand or gravel. Soil moisture, prebloom rainfall, tillage method, crop sequence, plant spacing, and soil fertility also may influence patterns of disease development in individual fields. Entire plantings often succumb simultaneously. Relative susceptibility of varieties apparently is influenced by such factors as drought resistance, heat tolerance, time required for seed development, and amount of seed produced *(5, 6)*. Among sorghum varieties, conditions required for predisposition to charcoal rot vary. Some short, exotic, high-yielding varieties are more likely to succumb to the disease than tall, native, low-yielding varieties under similar stress conditions.

The damage to sorghum plants from charcoal rot is usually not noticed until affected plants wilt or collapse. The fungus enters the plant through the roots and grows into the stem. Root attack at first appears as brown, water-soaked scattered lesions. It later darkens to a wet, black rot of the entire root *(27)*. In infected sorghum seedlings, the cortical tissue of the young stem at or near the soil level becomes flaccid, rots, turns reddish brown, and dies. Charcoal rot typically advances within the stalk from the crown upward in three successive phases: 1) general water-soaking of pith tissue; 2) intense pigmentation (red to black) of affected tissues; and 3) formation of sclerotia (hard, black, or dark brown bodies 1 to 2 mm in diameter) on the vascular remnants *(6)* (fig. 11). Affected plants lodge and tissues within the stalk break down and appear shredded. Under conditions favoring disease development, all three phases may be completed within a few days (fig. 12). If water-stress conditions are interrupted, the disease process may be inhi-

Figure 10. Bacterial soft rot on sorghum stalk. Note the hollow areas of gas pockets which separate this disease from insect damage.

Figure 11. Charcoal rot in sorghum stalks showing the progressive stages in tissue pigmentation and disintegration, both of which occur before sclerotial formation.

Figure 12. Terminal stage of charcoal rot showing distintegration of pith and parenchyma tissue.

bited or retarded, resulting in incomplete or incidental tissue damage. Sclerotial formation, physical collapse of stalk tissues, and invasion by other microorganisms may follow.

Tall, local sorghum and many forage sorghums are highly resistant to charcoal rot, but controlling charcoal rot in the exotic, high-yielding sorghums may be more difficult. Under borderline stress conditions, lessening the likelihood of stress during seed development may help. That can be done by spacing plants as far apart as practical, avoiding light or extremely well-drained soils, and fallowing or green-manuring every 2 or 3 years. If stress conditions are severe, however, such practices may not be effective because they tend to promote vegetative growth, thereby intensifying the moisture demands of the plant during its late development stages.

Growing varieties resistant to predisposing water-stress conditions is the best way to control charcoal rot.

Fusarium Stalk Rot

Fusarium stalk rot, incited by *Fusarium moniliforme* Sheld, is generally found in the same areas where charcoal rot occurs. Like charcoal rot, *Fusarium* stalk rot apparently requires some predisposing conditions for disease development as plants approach maturity. Unlike charcoal rot, however, *Fusarium* stalk rot usually is most damaging during cool, wet weather following hot, dry weather.

Fusarium stalk rot almost always is accompanied by extensive root damage. Under irrigation and heavy nitrogen fertilization, root damage may not cause any noticeable change in the above-ground appearance of the crop before stalk rot commences. The fungus also attacks maize, millet, and sugarcane. Morphologically, the fungus is the same as the one responsible for Pokkah boeng, but it probably is a different physiological race.

Root damage typically involves the cortical tissues first, then the vascular tissues of all roots. Newly formed roots may exhibit distinct lesions of various sizes and shapes. The rot is progressive, and older roots are often destroyed; the plant is left with little anchorage and is easily uprooted.

In dwarf sorghums, where leaf sheaths completely cover the stalks, there is little evidence of infection until the plants begin to wilt and dry *(28)*. In the taller varieties, with parts of the stalks not covered by leaf sheaths, discolored spots on the outside of the stalk at ground level indicate where the infection takes place. When stalks are split lengthwise, discoloration of the vascular bundles just inside the rind are apparent (fig. 13). The roots are also discolored, but the central pith core retains its original appearance. The interior of the stalk becomes water-soaked and the pith rots, leaving the vascular bundles as loose strands inside the outer cyclinder of the stalk.

Figure 13. Internal symptoms of *Fusarium* stalk rot.

Figure 14. *Fusarium* stalk rot on sorghum in Maroua, Cameroon. All the plants in the field have been killed, except for a few obviously resistant plants.

Fusarium stalk rot usually can be distinguished from charcoal rot because of its less pronounced pigmentation and disintegration of pith tissues, slower rot rate, and absence of sclerotia typical of charcoal rot. Where charcoal rot may destroy a field of sorghum in 2 or 3 days, *Fusarium* stalk rot may take several weeks (fig. 14). Coincident with increased *Fusarium* problems are several cultural practices suspected of contributing to the increase of the disease. Those cultural practices include high nitrogent fertilization, high plant populations, and continuous cropping.

Severe damage from foliage disease is most likely to occur under prolonged humid conditions. These humid conditions need not be directly correlated with periods of high rainfall. Intermittent low rainfall with dense cloud cover and little direct sunshine so that the leaves remain wet with dew for long periods can be more favorable to the development of foliage diseases than heavy rainfall followed by periods of bright sunshine that dries the foliage. *Sorghum* can sustain considerable damage to foliage without substantial yield losses, unless that damage occurs on the upper leaves at the time the grain is filling.

If individual sorghum foliage diseases appear by themselves on sorghum plants or in sorghum fields, identification of these diseases would be rather easy. However, environmental conditions favorable to the development of most sorghum foliage diseases encourage simultaneous development of all diseases that are present in the area at that time. Fortunately, foliage diseases of sorghum have certain characteristics by which they may be recognized in the field even though several diseases may be present on a single leaf. The size and shape of rust pustules, for example, remain constant regardless of how many other diseases are present. Similarly the shape and other identifying characteristics for zonate leaf spot, *Cerospora* leaf spot, oval leaf spot, sooty stripe, and so forth remain identifiable even after the death of the leaf or the plant on which they occur.

Some foliage diseases are more important than others. Those diseases that upset the physiology of the sorghum plant and result in its death, such as anthracnose, are obviously more important than leaf spotting diseases such as *Cercospora* leaf spot. However, sometimes a sorghum variety showing a dark lesion reaction when infected with bacterial stripe or *Cercospora* leaf spot will appear to be more injured than it actually is and will produce apparently normal yields.

Helminthosporium Leaf Blights

Leaf blight incited by *Exserohilum (Helminthosporium) turcicum* occurs sporadically on sorghum in West Africa. The disease is normally much more common and damaging to maize than to sorghum, although under certain environmental conditions it can severely damage susceptible sorghum varieties. Isolates of *E. turcicum* that attack maize generally are not damaging to sorghum. In test plots interplanted with maize and sorghum at Samaru, Nigeria, *E. turcicum* attacked only the maize in 1974. The following year the disease attacked only some susceptible sorghum varieties and not any of the maize, although the same varieties of each crop were grown each year.

Exserohilum leaf blight appears on leaves of susceptible sorghum plants as elliptical, grayish, or tan spots 30 to 200 mm long and up to 20 mm wide (fig. 15). Seedlings may be infected and, in severe cases, may die. Under conditions of prolonged high humidity the spots enlarge sufficiently to kill large parts of the leaves within 2 or 3 days. These dead areas wither quickly when dry weather returns so that the affected plants appear dried out. The centers of the spots are usually grayish or straw colored and, depending on the variety, have reddish-purple or tan borders. During humid weather, the fungus sporulates abundantly in dead leaf areas and spores are spread by wind to other plants. There the disease is quickly established if environmental conditions are favorable.

These spores on plant refuse carry over the disease from year to year.

Most of the sorghum varieties grown in West Africa are resistant to *Exserohilum turcicum*, but some of the introduced, short-season varieties may be severely damaged in certain areas.

Another leaf blight of sorghum is incited by *Helminthosporium maydis* (Nisikado) Shoemaker. This disease differs from the leaf blight incited by *E. turcicum* in that its lesions are smaller. These lesions are somewhat angular, because they may be limited by the leaf veins (similar to *Cercospora* leaf spot, but with a red or brown border and a grayish or tan center). These spots range in size from a few mm to 35 mm in length. *H. maydis* is not as damaging to sorghum as *E. turcicum,* probably because the fungus does not normally sporulate on sorghum and therefore does not spread on it as rapidly. *H. maydis* is primarily a disease of maize and is only a minor disease on sorghum when environmental conditions are right for it. Control measures for *Helminthosporium* leaf blights consist mainly of destroying plant refuse by clean cultivation or burning and controlling weeds which may serve as a source of inoculum.

Pokkah Boeng or Twisted Top

Pokkah boeng or twisted top is caused by the soil-borne fungus, *Fusarium moniliforme* var. *subglutinans,* which is limited to sorghum areas in West Africa where high humidity is prevalent during the growing season. Although the disease may be conspicuous on some sorghum varieties, losses usually are small. Pokkah boeng is characterized by deformed, folded, or discolored leaves near the top of the plant. In some cases, the leaves become so wrinkled they are unable to unfold properly, resulting in a plant with a ladder-like appearance (fig. 16). In extreme cases, infection may move from the leaves and sheath into the stalks, causing death of the tops. In mild cases, symptoms often resemble those of mosaic virus or yellow leaf blotch. Pokkah boeng can be differentiated from these two diseases by its characteristically wrinkled leaf bases and numerous small, transverse cuts in the leaf

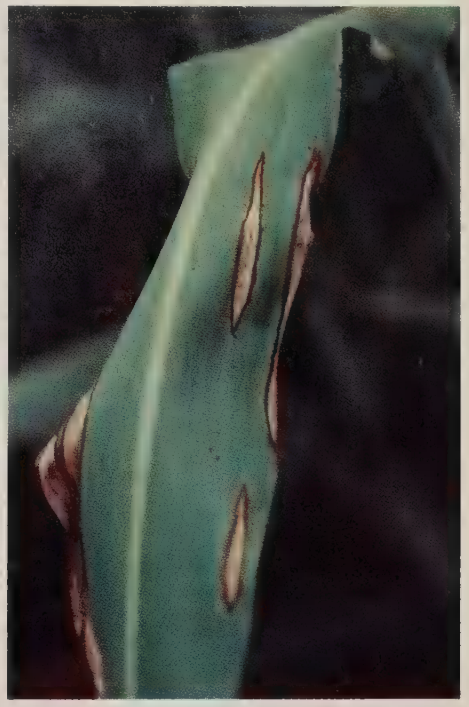

Figure 15. *Exserohilum turcicum* leaf blight on sorghum.

Figure 16. Pokkah boeng on sorghum plant preventing the leaves from unfurling properly, which gives the plant a ladder-like appearance.

Figure 17. Pokkah boeng or twisted top of sorghum.

margin (fig. 17). Sometimes the disease causes stalks to bend, and sometimes the stalks display "knife-cut" symptoms, which are narrow, uniform, transverse cuts in the rind that give the impression the tissue has been removed with a sharp knife (fig. 18). Because the lesions are covered by the leaf sheaths in sorghum, they may not be apparent when pokkah boeng leaf symptoms are present. Under conditions of physical stress, such as in windstorms, the stalks may break off along the "knife-cut" lesions so that the entire top of the plant is lost.

During prolonged wet weather, *F. moniliforme* var. *subglutinans* grows upward on the outside of sorghum stalks, may become temporarily established behind the leaf sheath or in the whorl. Metabolites produced by the fungus incite distortions in the plants. Later, when wet weather subsides, the fungus dries up and the plant resumes normal growth. There is some evidence that the fungus may also be seed transmitted. The fungus also affects maize, sugarcane, and millet.

There are presently no control measures for this disease.

Zonate Leaf Spot

Zonate leaf spot, incited by the fungus *Gloecercospora sorghi* Bain and Edgerton, is common on sorghum as well as on maize, millet, sugarcane, and numerous other grasses during humid periods in West Africa. The disease is conspicuous on sorghum leaves as circular, reddish-purple bands alternating with straw-colored or tan areas, which give a concentric, or zonate, pattern with irregular borders (fig. 19). The spots sometimes occur in semicircular patterns along the margins of the leaves. Ranging in diameter from 1 to 2 cm in early stages, to 3 to 7 cm in later stages, the spots may cover the entire width of the leaf. Both leaf blades and sheaths can become infected. In warm, wet weather, pink-to-salmon gelatinous spore masses form above leaf pores (stomata). High incidence of the disease on

Figure 18. Knife cut on sorghum stalks incited by *Fusarium moniliforme.*

plants in the seedling stage may result in severe defoliation, even death, of infected plants. Abundant spotting on leaves of older plants may cause premature destruction of foliage and poorly filled seed. The fungus may markedly reduce the amount of forage produced by sorghum-sudan hybrids.

The fungus overwinters as sclerotia that are formed readily within dead tissues of old leaf lesions as small, raised bodies in lines parallel to veins. Sclerotia are produced much more abundantly on millet and other grasses than on sorghum. Sclerotia germinate when the rains begin and give rise to spores (conidia) that infect the next crop *(3).* The fungus also may be carried on the seed.

Highly resistant varieties currently are not available. Breeding lines having good field resistance, however, are being identified and incorporated into desirable types. Likelihood of severe losses from this disease can be reduced by use of clean seed, crop rotation, and clean cultivation and burning to destroy residues of susceptible host crops.

Rough Spot

Rough spot incited by the fungus *Ascochyta sorghina* Sacc. is found only where sorghum is grown in the more humid areas. This conspicuous leaf disease is easy to identify by the sandpaper-like roughness caused by hard, black, raised fruiting bodies (pycnidia) of the fungus in the late stage (fig. 20). The disease begins as small, circular to oblong, light-colored spots within well-defined margins near the ends of leaves. Small, hard, black specks, the fruiting bodies of the fungus, develop in injured areas. As spots enlarge, they grow together so that the size of the diseased areas is highly variable. As the leaves mature, pycnidia may fall off or be washed from the leaves by rain so that only large areas of necrotic tissue remain. These lesions may resemble those of leaf blight. They can be separated from leaf blight in that there is no well-defined margin or halo associated with rough-spot lesions.

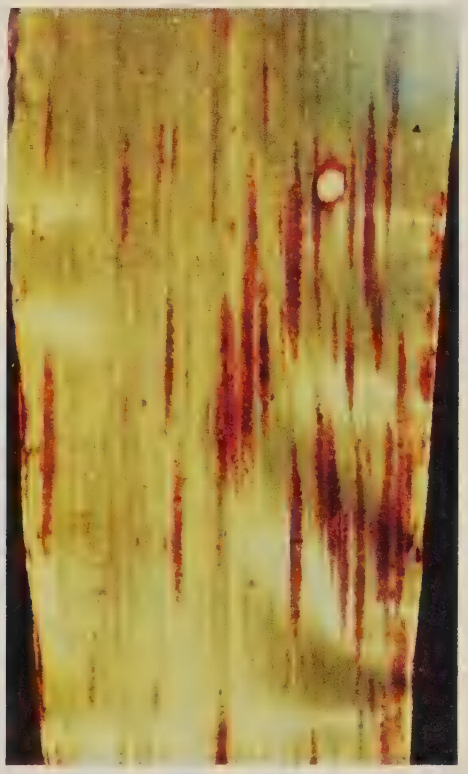

Figure 20. Rough spot on sorghum.

Figure 19. Zonate leaf spot on sorghum.

The disease is most severe in fields where sorghum has been grown during several successive seasons. The disease is spread mainly by airborne pycniospores during wet weather. The fungus is carried over from year to year on crop residues or sorghum and perennial weeds.

Losses due to rough spot are generally minor. Some sorghum varieties have high levels of resistance to rough spot. Crop rotation, burning of refuse, and clean cultivation to destroy plant residues help to reduce damage from this disease.

Cercospora Leaf Spot (Grey Leaf Spot or Angular Leaf Spot)
Grey leaf spot or angular leaf spot of sorghum incited by *Cercospora sorghi* Ell and Ev. is a conspicuous disease of rather minor importance. Grey leaf spot is probably the most widespread of all sorghum diseases in West Africa. The fungus attacks all forms of sorghum, maize, and numerous species of wild and cultivated grasses, which carry the fungus from year to year. The disease appears as small, circular to elliptical spots, usually dark purple or red, without a halo or differently colored margin. Later the centers may become tan to brown. Spots elongate until they are rather long and narrow (10 to 30 mm long by 2 to 3 mm wide). The leaf veins that limit them give them a long, rectangular appearance (fig. 21). As spots enlarge during humid weather, they may become covered with copious greyish mycelia of the fungus. Mycelia produce an abundance of conidia, so that during periods of high humidity, large areas of leaves may be covered with spots. Although all sorghum varieties are susceptible to grey leaf spot, the disease generally occurs late in the growing season after the crop is matured; little loss results.

Varieties of sorghum that are completely resistant to grey leaf spot are not available. Some varieties do, however, appear to be more resistant to the disease than others. Control measures are not warranted.

Rust
Sorghum rust incited by *Puccinia purpurea* Ckd. occurs throughout the more humid regions of West Africa. Rust appears on both surfaces of the leaves as small, raised, brownish pustules that are filled with powdery, brown spore masses (fig. 22). By gently rubbing raised pustules, one may easily differentiate between rust and other leaf spots. When the disease is severe, pustules may be so numerous that the entire leaf is destroyed. Once present in the crop, sorghum rust is spread by means of numerous repeating spores (uredospores). These spores may be carried for considerable distances by air currents and may be able to infect wet leaves of susceptible sorghum varieties and form new pustules in a few days. The means by which the fungus persists between crop seasons are not well understood. There is some evidence that the aecial, or carryover, stage may occur on *Oxalis* spp. It is more likely, however, that perennial weeds serve as the carryover host.

Normally, rust occurs late in the growing season after the grain has matured, and little loss results.

Varieties of sorghum resistant to rust are available. Those varieties should be grown in humid areas where rust may become a problem.

Sooty Stripe
Sooty stripe, incited by *Ramulispora sorghi* (Ell and Ev.) Olive and Lefebvre, commonly is found on leaves and sheaths of sorghum, sudangrass, johnsongrass, and broomcorn throughout West Africa. Oldest leaves are usually attacked first and most extensively. The disease first appears as small, water-soaked spots that may be colored with host pigment. These spots are normally elliptical (8 to 10 mm by 35 to 70 mm), but may fuse into irregular shapes. Spots begin as small, circular lesions that enlarge and lengthen rapidly during periods of humid weather so that lower leaves of plants may become covered with sooty stripes in 3 or 4 days. Affected leaves often turn bright yellow. Eventually black sclerotia form in abundance on the lesion surfaces; hence, the name sooty stripe (fig. 23). Heavy grey sporulation by the fungus often precedes the forming of sclerotia. Broad,

Figure 21. *Cercospora* leaf spot on sorghum leaves. Note the straight sides on the lesions.

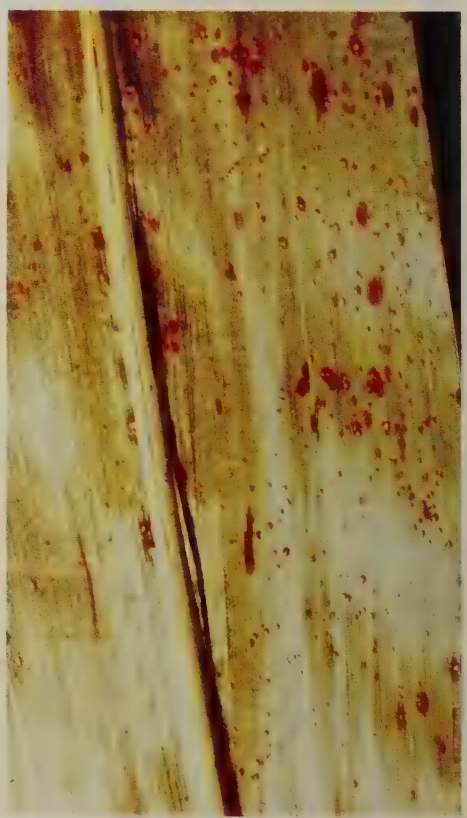

Figure 22. Rust on sorghum leaf.

yellow margins distinguish sooty stripe from leaf blight. Sclerotia of sooty stripe are small and irregular and tend to stick to the fingers like soot, in comparison to rough spot, which has smooth, round pycnidia that readily fall from the leaves. The fungus lives from one season to the next on crop residues.

Most sorghum varieties will sustain some damage from sooty stripe but generally are not affected early enough in the season to reduce yields. Sorghum varieties completely resistant to sooty stripe are not currently available. Clean cultivation and burning to destroy crop refuse, along with crop rotation and seed treatment, aid in the control of the disease.

Oval Leaf Spot

Oval leaf spot, incited by *Ramulispora sorghicola* Harris (14), is one of the newer sorghum diseases in West Africa. Oval leaf spot was first described in Nigeria in 1960 and has since worked its way into most of West Africa. The disease appears on all leaves of sorghum plants during humid weather as small, water-soaked areas with an indistinct red or brown margin. Spots become elliptical and grow up to 7 by 3 mm (mostly 3 to 4 by 1 to 2 mm) with a conspicuous dark red, brown, or tan margin up to 1 mm wide (fig. 24). Centers of the spots are a bleached straw or tan color. As spots age, a few, small, black sclerotia sometimes form in the tan straw-colored centers.

Oval leaf spot attacks sorghum plants at all stages of growth. Heavy infections sometimes occur in fields that are cropped to sorghum every year. Fortunately, however, the disease does not cause premature death of leaves and losses are not serious. The fungus is carried over from year to year on diseased leaf tissue. Clean cultivation and destruction of crop refuse by burning along with crop rotation aid in controlling oval leaf spot.

Figure 24. Oval leaf spot on sorghum leaves.

Figure 23. Sooty stripe on sorghum.

Downy Mildews

Sorghum downy mildew, incited by *Peronosclerospora sorghi* Weston and Uppal, and green ear, incited by *S. graminicola* [Sax.] Schroet., are found sporadically on sorghum in West Africa, generally on introduced short sorghums or on early volunteer plants. The tall, native varieties either have resistance to these diseases or are planted late enough to escape them.

Sorghum Downy Mildew—The most conspicuous symptom of the systemic phase of sorghum downy mildew is the appearance of vivid green and white stripes on leaves when plants begin jointing (fig. 25). Plants systemically infected with downy mildew frequently fail to head. Another symptom of downy mildew is the appearance of down on the undersides of mottled but unstriped leaves during humid weather (fig. 26). This down or fuzz is made up of asexual fruiting bodies (conidia and conidiosphores) of the fungus. Conidia are spread by wind and cause infection of other plants in the field.

Foliar infection produced from conidia somewhat resembles mosaic symptoms in that leaves are mottled and yellow. Downy mildew, in many instances, however, can be distinguished from mosaic by the presence of white downy conidiosphores during humid weather. Foliar infection may repeat itself during a growing season so that additional plants may become infected. If foliar infection occurs early in the growing season, infected plants may develop systemic symptoms later in the season. These plants may not produce seedheads.

Still another symptom of downy mildew, the local infection phase, is the appearance of small (1 to 2 mm), red to brown, circular spots on the upper sides of leaves late in the season. These spots may be localized on a single portion of the leaves or may be scattered all over the foliage. These spots develop from conidial infections that do not become established well enough to produce down or become systemic. Most sorghum varieties in areas where downy mildew is present will show some of these local infection symptoms but will not suffer any apparent injury from them. Even resistant varieties will show this symptom without ill effect.

As the systemically infected leaves shred, oospores (sexual spores) are released from the white striped areas of the leaf into the soil (fig. 27). These oospores may remain viable in the soil over a period of several years. When sorghum is planted in an infested field, these oospores can infect the young seedlings before they emerge from the soil. Those infected seedlings that emerge will show a chlorotic mottle. Infected plants that do not die at a young age generally develop systemic symptoms and the disease cycle is repeated. Sorghum downy mildew is not seed transmitted, but the fungus can be spread on trash mixed with seed.

Figure 25. Downy mildew of sorghum showing the white-striping characteristics of sorghum downy mildew.

Figure 26. Downy mildew showing profuse production of down under conditions of high humidity.

Figure 27. Sorghum plant infected with downy mildew causing leaf shredding, which occurs along the white stripes.

Varieties of sorghum resistant to downy mildew are available. These should be grown in areas where downy mildew is prevalent.

Green Ear—Green ear, which is the most important disease of millet in West Africa, occurs on young sorghum only during extended periods of extremely high humidity. *S. graminicola* causes a downy mildew on the leaves similar to that of *P. sorghi*. It also causes a condition in which the head is transformed completely or partially into a loose, green head composed of a mass of small, twisted leaves known as "green ear" *(27)*.

Green ear is sporadic in occurrence and of little economic importance on sorghum. Control measures are not warranted.

Mosaic

Mosaic, incited by the sugarcane mosaic virus (1), is found on sorghum and maize in some of the more humid areas of western Africa The disease is generally confined to the vicinity of mosaic-infected sugarcane plants *(Saccharum officinarum* L.) or Elephant grass (*Pennisetum purpureum* Schumac.), which harbor the virus from year to year.

Mosaic normally appears on the upper two to three leaves of sorghum plants as an irregular mottling of green and light green areas, often interspersed with longitudinal white or light yellow streaks *(1)* (fig. 28). On some varieties, however, the disease sometimes appears as a brilliant red-mottled streaking of the upper leaves or as mottled necrotic lesions on the upper leaves (fig. 29). This is known as the red leaf phase. These spots are not watersoaked like bacterial-induced spots. The effect of mosaic on sorghum plants will vary considerably depending on variety and time of infection. Plants infected at an early stage of growth may be severely stunted and often fail to set seed. Grain yield may be reduced when compared with plants infected at or near maturity.

In the field mosaic is transmitted from diseased sugarcane plants or elephant grass to sorghum by several species of aphids. These aphids feed on the diseased plants and then move to healthy plants to which they spread disease with their piercing mouth parts. Sugarcane mosaic virus is nonpersistent in the vector, which means that aphids can transmit the virus for only a short time (about 1 hour) after they leave the diseased plant.

Sugarcane mosaic is not an important disease of sorghum in West Africa because of the failure of the disease and the vectors to survive the dry season. Control measures are not warranted.

Figure 28. Sugarcane mosaic on sorghum showing the mild or green reaction.

Figure 29. Red leaf phase of sugarcane mosaic virus on a sorghum variety that gives the red color reaction to disease.

Phoma/Phyllosticta Leaf Spots

Leaf spots of sorghum, incited by *Phoma insidiosa* Tassi and *Phyllosticta sorghiphila* Sacc. *(25)*, can be recognized on sorghum leaves as subcircular to irregular-shaped, tan spots 1 to 3 cm or larger with red or

Figure 30. *Phoma/Phyllosticta* leaf spot on sorghum.

brown margins (fig. 30). Sometimes the lesions begin at the tips or margins of the leaves *(27)* and appear elongated. When numerous, spots may fuse, giving the leaf a net-like or reticulate appearance. Spots, however, retain their original margins even after the entire leaf is covered. Pycnidia develop as tiny, black dots on dead tissue in groups of lines between veins. These two fungi also occur on seeds and glumes of sorghum where they are carried over from season to season. They may also survive on dead plant material and on weeds. Infection takes place from spores liberated from the pycnidia during wet weather.

Losses caused by these leaf spots on sorghum are not of economic importance. However, when *Phoma Phyllosticta* is carried on seed it may reduce germination and cause seed rot and seedling blight, which may be of considerable economic importance *(19).* The use of clean seed or seed treated with seed dressings (seed treatment) gives satisfactory control.

Bacterial diseases of sorghum are more numerous and widespread than formerly believed. The role that bacteria play in the sorghum disease picture in West Africa is not completely known. One may be sure that all the bacterial diseases of sorghum in Africa have not yet been identified. Some bacteria may possibly protect sorghum plants against infection by certain fungi. Giha *(13)* in Nigeria showed that by treating wheat seed with a bactericide the percentage of seed germination was reduced by up to 70 percent and the level of foot-rot in seedlings from the treated wheat seed was increased by the same percentage.

Bacteria responsible for sorghum diseases are carried over from season to season on seed, on infected plant material in soil, or on local weed grasses. In the field bacteria are spread from diseased to healthy plants by wind, wind-splashed rain, insects, and cultivation when the foliage is wet. Bacterial diseases are controlled by using clean seed, crop rotation, clean cultivation, and burning to destroy plant residues.

Sorghum varieties differ in susceptibility to individual bacterial diseases, but varieties distinctly resistant to all bacterial diseases have not yet been developed. Sorghum plants respond to tissue injury by producing color pigments characteristic of the variety. Some varieties, when injured, will produce a reddish-purple pigment; others, red or brown pigment; and still others, a tan or yellow pigment. Since the dark pigments are more conspicuous than the light tan or yellow ones, farmers and other agricultural workers have a tendency to give greater importance to dark-colored lesions and to overlook or minimize damage done from tan or light-colored lesions.

Bacterial Stripe

Bacterial stripe, incited by *Pseudomonas andropogoni* (E. F. Smith) Stapp. *(8),* is commonly found on sorghum throughout West Africa. The disease is characterized by tan to brick-red to dark purplish-red stripes 3 to 8 mm wide (fig. 31). These are generally restricted to intervenial areas of apical portions of lower leaves. Sometimes the entire plant may be affected. Ranging from less than 25 mm to the total length of the leaf blades, stripes sometimes fuse so that large areas of the leaf are affected. The lesions, which are not watersoaked, are often somewhat irregular in shape with the ends blunt or prolonged into long, jagged points. A slime or bacterial exudate may be found on the underside of affected portions of the leaves and along the leaf margins, which is readily washed off by rain. The bacterial exudate may be best seen in sunlight reflecting on thin, glistening scales the same color as the lesions. The shape of the stripes is generally the same on all sorghum varieties, but the color varies. Some varieties always show a purplish or blackish-red reaction, whereas other varieties always show a tan color (fig. 32.)

Although conspicuous, especially on varieties that give the dark reaction, losses caused by bacterial stripe are generally not severe. Sometimes the disease is overlooked in some sorghum varieties that give the tan or light reaction. Plants on which the entire leaf is covered by the disease will generally still produce a good seed head. Grain from these plants, however, should not be used for seed, as the bacterium will be carried over on them.

Control of bacterial stripe at present consists of using seed from plants that do not show disease symptoms in the field, clean cultivation, and burning to destroy plant refuse and crop rotation.

Bacterial Streak

Bacterial streak, incited by *Xanthomonas holcicola* (E.C. Elliot) Starr and Burkh *(8),* is less commonly found in West Africa than bacterial stripe, but when present, it may do more damage to individual plants.

Figure 31. Bacterial stripe on sorghum leaf.

Figure 32. Bacterial stripe on sorghum leaves showing three distinct color reactions. The leaves are equally infected.

Bacterial streak appears as irregular, translucent streaks about 3 mm wide by 25 to 30 mm or more long, which may develop on plants any time between the seedling stage and near maturity. At first no color is evident except the light-yellow, beadlike drops of exudate standing out on the young streaks. Later, narrow, red-brown margins or blotches appear in the streaks; after a few more days, the streaks are red throughout and no longer appear watersoaked or translucent. Parts of streaks may broaden into elongated oval spots with tan centers and narrow, red margins (fig. 33). When numerous, streaks may join to form long, irregular areas covering much of the leaf blade. At that advanced stage, dead tissue with dark, narrow margins forms between reddish-brown streaks, and the bacterial exudate dries to thin, white, or cream-colored scales.

Bacterial streak may be distinguished from bacterial stripe *(Pseudomonas andropogoni)* in that lesions of bacterial stripe are never watersoaked, are uniform in color, and never take the form of oval spots with tan centers and red borders. Control of bacterial streak is essentially the same as that for bacterial stripe.

Bacterial Spot

Bacterial spot, incited by *Pseudomonas syringae* Van Hall (8) is found throughout West Africa on sorghum, millet, numerous grasses, and several unrelated crop plants such as banana (Musa spp.), beans *(Phaseolus* spp.), and tomato *(Lycopersicon* spp.). On sorghum, spots appear first on lower leaves as the plants approach maturity. Spots, which occur on any part of the leaf, usually are circular to irregularly elliptical and are from 1 to 8 mm in diameter. Appearing dark green and watersoaked at first, they turn red in a few hours. They soon

Figure 33. Bacterial streak on sorghum leaves. Note the wide areas with tan centers which separate bacterial streak from bacterial stripe.

become dry and light-colored in the center, retaining a red border (fig. 34). Most small lesions are red throughout, having tiny, somewhat sunken centers, but the colors bordering the lesions varies from red to dark brown in some varieties. Frequently, the spots are so numerous that they coalesce into large diseased areas and the whole leaf dies.

Bacterial spot is more difficult to identify in the field than the previous two bacterial diseases, especially in the later stages when spots become dry. Several fungus leaf spots, which are superficially similar to them, may occur on the same plant. Oval leaf spot *(Ramulisphora sorghicola)* and young infections of *Cercospora sorghi* resemble bacterial spot but may be separated from it. Oval leaf spot, and *Cercospora* leaf spots occur fairly randomly on the surface of infected leaves, because these infections come from individual airborne spores which land on leaves. Bacterial spot lesions tend to be concentrated on one portion of the leaf, because these lesions originate from bacteria that migrate from a central infection source.

Control of bacterial spot is essentially the same as that for bacterial stripe.

Figure 34. Bacterial spot on sorghum leaves.

Red Stripe and Top Rot

Red stripe, incited by *Xanthomonas rubrilineans* (Lee *et al.*) Starr and Burkholder, is normally a disease of sugarcane, but it will also attack sorghum in West Africa. Red stripe appears on the basal portions of sorghum leaves as fine, irregular stripes with a watersoaked appearance. Later, these stripes turn red or brownish purple. They range from 15 to 40 cm long and 1 to 3 mm wide, with irregularly wide areas or spots 4 mm wide. Split stalks of severely diseased plants show a watersoaked red cylinder reaching from the dead apical bud to the base of the stalk. In cross section, diseased stalks show a red circle around a watersoaked core. Tops of diseased plants die, but lower buds may resprout.

Only individual plants infected with red stripe have been observed in Nigeria and Senegal. Control measures are not warranted.

Bacterial Sun Spot

Bacterial sun spot, incited by *Pseudomonas* sp. *(34).* is found in eastern Senegal. The disease is characterized by conspicuously isolated, circular to elliptical, dark red or brown spots with tan or light centers on the upper leaves of sorghum plants (fig. 35). Spots range in size from 4 to 23 mm in diameter with some larger ones being 19 mm wide by 50 mm long. An abundant, reddish-brown bacterial ooze resembling droplets of blood is found on undersides of spots. Bacterial sun spot generally attacks plants just before flowering. The exact nature of the method of infection is not known. Airborne infection is indicated, because when the disease appears, almost all plants in the field have sun spots.

Figure 35. Bacterial sun spot on sorghum leaves.

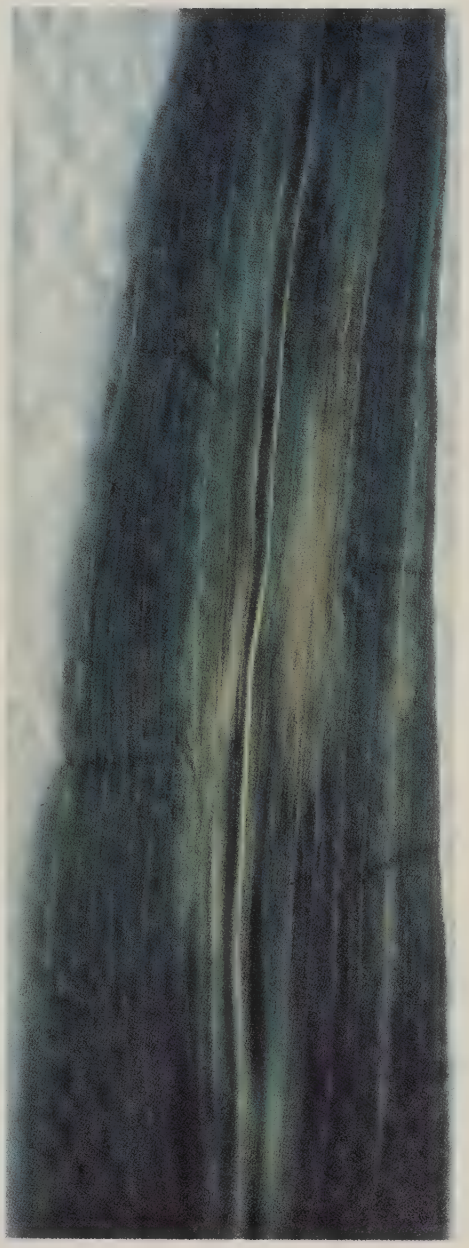

Figure 36. Yellow leaf blotch on sorghum leaves.

The disease is also found on sugarcane, but spots are smaller and bacterial ooze is less conspicuous than on sorghum.

Sun spot, although conspicuous on those sorghum varieties that show a dark red reaction, does little injury to the plant. Control measures are not warranted.

Yellow Leaf Blotch

Yellow leaf blotch, incited by *Pseudomonas* sp. (32), is a recently identified bacterial disease of maize, millet, and sorghum found throughout West Africa. The disease has been observed in Cameroon, Ghana, Niger, Nigeria, and Senegal. On sorghum leaves, the disease appears as scattered, cream-yellow to light-beige lesions 25 to 35 mm long by 8 to 10 mm wide (fig. 36). Some lesions are slightly water-soaked and have a tendency to follow veins. Young plants infected at the 2- to 3-leaf stage are sometimes severely stunted or killed.

In some respects, symptoms of this bacterial disease resemble those incited by the parasitic weed *Striga hermonthica*. However, symptoms of both can be readily

separated in the field. The bacterium attacks plants at an early age starting at the seedling stage, whereas *Striga* symptoms usually begin to appear when sorghum plants are about to bloom. Lesions incited by bacteria are cream-yellow to light-beige and generally distinct, with a tendency for runners to follow veins. With *Striga* injury, however, leaves become diffusedly bleached and then watersoaked but do not take on the distinct yellow color of the bacterial lesions. When areas of leaves from *Striga*-affected plants wither and die, they have an overall bleached appearance.

Symptoms of the disease have also been observed on johnsongrass *(Sorghum halepense)*, wheat *(Triticum aestivum)*, and several unidentified grasses in Nigeria. The economic importance of yellow leaf blotch has not yet been fully determined. Mature plants of maize, millet, and sorghum can apparently produce a normal crop when infected after the seedling stage. Young plants of sorghum and millet, when infected early, may be killed or severely stunted.

An interesting aspect of yellow leaf blotch is that on sorghum, where lesion color is normally influenced by plant variety, yellow leaf blotch lesions are uniformly yellow in color even on those varieties that produce red-pigmented disease lesions.

Control methods have not yet been developed.

Smut Diseases

There are four smut disease found on sorghum throughout West Africa: Covered kernel smut *(Sphacelotheca sorghi)*, loose kernel smut *(S. cruenta)*, head smut *(S. reiliana)*, and long smut *(Tolyposporium ehrenbergii)*. The major symptom of these smuts is the development of smut sori, or black spore-filled pustules or sacks, in place of individual florets or of the entire sorghum head. During sorus development, vegetative fungal growth is converted to dark-colored spores (teliospores), which are contained in masses within a fungal-host membrane (peridium). Generally little damage is observed on the vegetative plants, except in some rare instances of foliar infection. Plants of some of the taller sorghum varieties may be severely stunted when systemically infected with certain smuts. Little stunting is noticeable on plants of dwarf varieties.

It is important to know the life cycle of a particular smut if satisfactory control is to be obtained. Loose kernel smut, covered kernel smut, and head smut initiate infections during the seedling stage of growth. These infections are carried into the floral primordia as the plant develops. Just before exsertion of heads, the smut fungi grow very rapidly in developing floral parts until space and nutrients are exhausted; then sori develop. Long smut infection takes place when sorghum plants begin to flower. Covered kernel smut and loose kernel smut are seedborne and can be effectively controlled with seed dressing. Head smut and long smut are soilborne and cannot be controlled with seed dressing. There are varieties of sorghum, however, that are resistant to long smut and head smut.

Covered Kernel Smut

Covered kernel smut, incited by *Sphacelotheca sorghi* (LK) Clint, can be identified by the generally smooth, spherical to cylindrical sori that develop in place of the ovary or stamens of individual florets (fig. 37). Florets may support a single sorus in place of the ovary or fused sori in place of both ovary and stamens. Fused sori often are lobed according to the number of floral parts included.

The number and distribution of smutted florets in a panicle varies. Sometimes all florets may be smutted, sori may be restricted to one portion of a panicle, or they may be scattered throughout the panicle. Sori may be oval, conical, or cylindrical. Some are small enough to be concealed by glumes. Others may be a centimeter long. They may be white, grey, or brown. Peridia of sori are variable in thickness; some rupture rather easily, but others persist until threshing. Contamination of seed during threshing is the normal means of spore carryover from one season to the next.

Infection occurs in the early stages of seedling development. The fungus becomes established in primordial tissues of developing shoots, and spores are produced in kernels and adjacent floral tissues. Soilborne spores are not considered important in the disease cycle.

Presence or absence of sori in heads of main stalks, auxiliary branches, or tillers depends on patterns of early infection. Main heads may escape infection if the uppermost cells of the apical meristem are not invaded. Secondary heads likewise reflect the incidence of infection in other cells of the apical meristem.

Sometimes plant height, internode number, stalk diameter, and leaf width may be reduced in some varieties infected by *S. sorghi*. Although at least five distinct races of *S. sorghi* have been identified, control of this disease is easily obtained with seed dressings.

Loose Smut

Loose smut, incited by *Sphacelotheca cruenta* (Kuhn) Potter, is somewhat similar to covered kernel smut in appearance and in transmission by seed. Loose smut can be separated from covered kernel smut in that the sori peridia of the former rupture soon after the emergence of heads. A long, columella (the deformed remains of florets) protrudes from the glumes after the spores have been dispersed (fig. 38). The glumes are usually elongated. Sori commonly develop on the rachis and its branches, sometimes on glumes, and occasionally on pedicles and stalks. Plants attacked by *S. cruenta* invariably head prematurely and produce few or no healthy heads and little, if any, grain. Plant height is markedly more reduced by *S. cruenta* than by *S. sorghi.* Excessive tillering is also encouraged.

Figure 37. Covered kernel smut on sorghum.

Figure 38. Loose smut on sorghum.

The primary method of dissemination of loose smut is by contamination of seed during threshing. However, under suitable conditions, sorghum heads may become infected by airborne spores late in the season. This type of infection is generally localized and of little consequence. Loose smut is easily controlled with seed dressings *(20).*

Head Smut

Head smut, incited by *Sphacelotheca reiliana* (Kuhn) Clint, differs from the other sorghum smuts in that the entire sorghum panicle or part of it becomes incorporated into a single, large sorus (figs. 39 and 40). Parts of an infected panicle that are not included in a sorus usually exhibit blasting or proliferation of individual florets. Young sori of head smut are sometimes eaten by certain people in West Africa before Peridia erupt.

Sori also may occasionally develop on foliage and culms in some sorghum varieties. The sorus has a thick, whitish peridium that ruptures readily after exsertion, and the black mass of teliospores gradually falls to the soil. Dark filaments several centimeters long, which are remnants of the vascular tissues, remain after the telispores have been shed. These filaments in the single sorus separate head smut from all other sorghum smuts. Spores from smutted heads, scattered by wind and rain, survive in the soil over the dry season in a dormant state. Sorghum seedlings are attacked at an early stage of growth and the infection becomes systemic. Usually the disease is not recognized until the first smutted inflorences emerge and the sorus appears. Some stunting may occur on some of the tall, local varieties when infected with *S. reiliana*. The fungus attacks several other grasses, including maize, johnsongrass, and Sudangrass.

Figure 39. Head smut on sorghum showing the white peridium.

Figure 40. Head smut on sorghum showing numerous long filaments within a single sorus.

Head smut cannot be controlled by the presently available chemical seed treatments, because the fungus is soilborne and can attack seedlings after they have grown out of the zone of protection offered by the seed protectant. There are sorghum varieties that are resistant to head smut and should be grown in those areas where head smut becomes a problem. Possibility of development of new pathogenic races on currently resistant sorghum varieties cannot be ruled out.

Long Smut

Long smut, incited by *Tolyposporium ehrenbergii* (Kuhn) Pat., is found in the drier regions of the Sudan and Sahel savannah zones of West Africa. Long smut can be identified by the large, long, cylindrical, slightly curved sori (2 to 5 cm long by 6 to 10 mm in diameter) that are formed on individual florets in sorghum heads (fig. 41). Sori are enclosed in a relatively thick, grey to brown peridium composed of fungus tissue. Sori of long smut resemble those of covered kernel smut but are much larger and have numerous, dark filaments running throughout the length of the sorus. Long smut sori do not have a columella as do loose smut sori. The spores are dispersed when the sorus ruptures at the tip or falls to the ground when the crop is harvested. Spores survive in and on the soil during the dry season. Infection is by airborne spores. Spore balls consisting of numerous telispores are picked up by winds that precede rainstorms and are deposited on flag leaves of sorghum plants *(17).* The ensuing rain then washes spore balls into the boot where spore germination occurs and infection of florets takes place.

Long smut is more serious in dry years than in normal wet years. In drought years the top soil becomes loose and dry, facilitating the uplift of soil particles and long smut spore balls by winds. More long smut sori occur at the base of the panicle than at the top because spore balls, washed by percolating rain water, collect in the tighter, lower end of the flag leaf sheath where water is also trapped. The high humidity in the boot encourages germination of the teliospores whose resultant sporidia come in close contact with the young florets still enclosed by the boot.

Chemical control of long smut is not practical, because large amounts of chemicals would have to be applied to entire sorghum fields before it was known how extensive infection would be in any particular year. The use of varieties resistant to long smut offers promise for the control of this disease.

Figure 41. Long smut on 'Short Kaura' sorghum.

Figure 42. *Fusarium* head blight on sorghum grain.

Fusarium Head Blight

Fusarium head blight, incited by *Fusarium moniliforme* Sheld., can be a serious problem on some of the dwarf, exotic, short-season sorghums in West Africa. *(33)*, especially if they flower during extended periods of heavy rains and high humidity. The disease is characterized by death of several to all of the florets in seed heads. When the disease is severe, the entire seed head may be covered with a copious cream to pinkish-tan fungal growth (fig. 42.) In extremely severe cases, heads of all plants in a field may be killed. If the panicle is split lengthwise, a red-brown to black discoloration is evident in the upper portion of the peduncle and extends into branches of the head. Sometimes discoloration may extend throughout the peduncle and into upper internodes of the stalk, in which case the rind may also be discolored. In severe cases, breakover of peduncles may occur.

Fusarium head blight may be separated from red rot and peduncle breakage incited by *Colletotrichum graminicola*. Discoloration in head blight is uniform throughout, whereas discoloration in red rot is interspersed with discrete, white areas.

The mechanism of infection and penetration by the fungus is not fully understood. It is suspected that mycelia of the fungus, which lives in the soil, grow up along the outside of the stalk on the waxy bloom during extended periods of wet weather. Alternatively, the fungus may infect the head through airborne conidia. Penetration may occur through cracks or insect wounds in the rind or peduncle, rachis, or panicle branches. Sorghum varieties with dense, compact heads are more prone to attack by head blight than are varieties with loose, open heads. Most of the tall, local sorghum varieties are resistant to *Fusarium* head blight. Some of the early, exotic sorghum varieties show excellent resistance to head blight and should be grown where short-season sorghums are recommended.

Ergot or Sugary Disease

Ergot or sugary disease of sorghum, incited by *Spacelia sorghi* McRae (probably an undetermined species of *Claviceps*) *(11)*, is primarily found on male-sterile sorghums in West Africa. As a rule, the disease is not serious. The most conspicuous symptom of ergot on sorghum is the production of a sticky, sugary solution from the ovaries of young florets soon after they open during periods of humid weather. This sugary secretion attracts numerous insects, especially flies, which aid in the spread of the disease (fig. 43). The sugary liquid first appears as pinkish drops and later becomes discolored because of growth of saprophytic fungi. If plentiful, the exudate converts the head into a sticky, black mass.

Sclerotia, as seen by the author, develop as medium, soft-pink, chalky, tumescent cylinders 1.5 to 1.7 mm by 4 mm long with blunt ends formed within the glumes. These pinkish sclerotia, which crumble when handled, are easily separated from glumes. In India sclerotia are reported to be 0.4 to 0.6 cm by 1.00 to 2.5 cm long, hard, cylindrically elongated, slightly curved, horn shaped *(23, 27)*.

Infection is through airborne conidia or insect borne conidia, sometimes between the time the flowers open and fertilization occurs. Native grasses probably serve as the primary source of infection, as germination of sclerotia has not been reported *(27)*. Because male-sterile sorghum varieties are susceptible, ergot or sugary disease may become a problem in hybrid seed production in West Africa. Control measures are not warranted at present.

False Smut

False smut, incited by *Cerebella sorghi-vulagris* Subram., is generally found as a secondary saprophyte associated with ergot or sugary disease *(Sphacelia sorghi* McRae). False smut can be easily identified by the small (1 to 3 mm), hard, dark purple to black stroma which replace entire kernels within the florets of sorghum heads (fig. 44). These stroma or hard bodies have convolutions on them like the striations on the surface of a brain, from which the name *Cerebella* is derived. There is evidence that false smut is not a true disease of sorghum but is actually a saprophyte on the honey dew resulting from sugary disease. There is some evidence that false smut may retard or inhibit development of the *Sphacelia*.

Since ergot or sugary disease of sorghum is normally restricted to male-sterile sorghum varieties, false smut in turn is confined mainly to these male-sterile varieties. Although conspicuous, false smut does little, if any, damage to sorghum; control measures are not warranted.

Figure 43. Ergot or sugary disease on male-sterile sorghum.

Figure 44. Cerebella or false smut on ergot on male-sterile sorghum.

Several species of *Striga (S. asiatica* (L.) Kuntze, *S. densiflora* (Benth.) occur in West Afica. These are all true parasites in that they must obtain some portion of their nutrition from a host plant. *S. asiatica, S. densifolia*, and *S. hermonthica* normally attack sorghums, maize, millet, and numerous wild and cultivated grasses, but not broadleaf plants. *S. gesnerioides,* on the other hand, normally attacks only broadleaf plants, such as cowpea and tobacco, but not the grasses. Sometimes confusion arises because of reports of *S. asiatica* or *S. hermonthica* on groundnuts or some other broadleaf crops. However, close examination will show that these two Strigas are actually parasiting some rather inconspicuous grass in the field.

In West Africa, *S. hermonthica* is by far the most important of these *Strigas.* It is probably the most important single disease pest of maize, millet, and sorghum in West Africa.

Striga hermonthica (giant witch weed)

Striga hermonthica, or giant witch weed, also called Wuta Wuta or Kuduji in Hausa, can be distinguished from the other *Strigas* by its large size (0.25 to 0.9 m tall with leaves 3 to 9 cm long by 3 to 7 mm wide) and large, showy, purple to purplish-pink flowers (fig. 45). The flowers have a corolla tube up to 29 mm long and lobes up to 12 mm long. Seed capsules are lanceolate, cylindrical, 3 to 4 mm in diameter by 8 to 11 mm long, and subtended by linear lanceolate bracts 4 to 6 mm long. When pulled and allowed to dry, *S. hermonthica* plants turn grey-green to black in 2 to 3 hours.

Striga hermonthica is very abundant on maize, millet, and sorghum in the Guinea savannah, the Sudan savannah, and the Sahel zones. It is less abundant in the southern Guinea zone and is rare or absent in the forest regions of West Africa. In the well-manured land close to villages, heavy infection prevents effective grain production *(21).* Production then has to be done at distances inconvenient to the village. In some areas, it is no longer possible to grow the most palatable and productive sorghum varieties because they are too susceptible to *Striga* attack.

Figure 45. *Striga hermonthica* plants in bloom.

Infection occurs when sorghum is planted in fields infested with *Striga* seed produced on crops from previous years. *Striga* seed in close proximity to young sorghum roots are stimulated into germination by an exudate produced by the sorghum root. The germinating *Striga* seed produces a root-like growth (haustorium) that penetrates the sorghum root and attaches the *Striga* to the sorghum. The parasite and host roots join at a small, bulbous swelling on the sorghum. Further haustoria may develop from a single *Striga* plant and may make parasitic contact with roots at many different places *(27),* or numerous *Striga* plants may attack a single sorghum plant.

Figure 46. Young *Striga* plants emerging under a very susceptible sorghum variety.

Figure 47. Heavy infestation of *Striga hermonthica* on cropped sorghum near Maroua, Cameroon.

Once attached to the sorghum plant, the *Striga* obtains all of its water, minerals, and other nutrition from the host. The *Striga* plants makes all of its growth underground until about the time that the sorghum begins to head. Then the *Striga* grows very rapidly and begins to emerge from the soil (fig. 46). It is at this time of *Striga* emergence or just before it that the damage from the *Striga* becomes most apparent. In order to produce its above ground organs, *Striga* draws nutrients from the sorghum plants faster than the sorghum plant can produce them. As a result, the host plant quickly declines or collapses. Until this time the only symptoms of *Striga* on the above ground portions of the sorghum are a general unthriftiness and a diffuse, fine, pinpoint spotting or mild bleaching of the sorghum leaves. At or just before *Striga* emergence, the lower leaves of the sorghum plants become water-soaked and bleached and then wither and dry up all within 3 to 5 days. A field infested with *Striga* may appear to be singed by fire or suffering from severe drought injury (figs. 47 & 48). Some susceptible sorghum varieties, when heavily infected, cease further development at this stage or die.

The amount of injury that a sorghum variety suffers from *Striga* attack depends on the tolerance of the variety to *Striga* and on the number of *Striga* plants parasiting individual sorghum plants. In very heavily infested fields, there may be 40 or more *Striga* plants on each sorghum plant.

Figure 48. Heavy *Striga hermonthica* infection on sorghum intercropped with groundnuts near Zaria, Nigeria.

The emerged *Striga* plants grow very rapidly and begin to produce flowers 20 to 30 days after emergence. Seed pods mature 20 to 30 days later. Seed of *S. hermonthica* are very small and appears to the naked eye as fine, greyish-black dust. Somewhere between 50,000 and 1 million seed are produced per *Striga* plant. These seed may be scattered considerable distances when the pods are ruptured. They may be carried considerable distances by wind, especially dust storms, and by heavy rains or irrigation water. In addition, *Striga* seed may be disseminated on clothing and implements of farm workers, by livestock, or on sorghm stalks used as building material. *Striga* seed may remain dormant for several years in the soil until they are stimulated into germination by the exudate produced by a suitable plant.

Striga asiatica

Striga asiatica is far less commonly found on sorghum in West Africa than in *S. hermonthica*. They both attack the same range of plants, but for some unknown reason, *S. asiatica* has not become very widely established or as important in West Africa. *S. asiatica* is similar in growth and form to *S. hermonthica* but may be easily differentiated from the latter by its smaller size and bright red flowers. *S. asiatica* is normally from 10 to 20 cm tall with linear leaves 1 to 3 cm long. Flowers have a corolla tube about 1 cm long with lobes 3 to 6 mm long. Flowers are normally bright red, but may be yellow or red with a yellow throat.

Striga densiflora

Striga densiflora is very rarely found in West Africa. It is similar to *S. asiatica* in size, but can be separated from it by its very small, white flowers.

Striga gesnerioides

Striga gesnerioides is sometimes found on cowpeas in fields intercropped to sorghum and cowpea. *S. gesnerioides* has pink to purple flowers similar to *S. hermonthica* but the plant is much shorter (20 cm). *S. gesnerioides* can be easily separated from other *Strigas* because it produces several (5 to 15) stems from a single, large (20 to 45 mm), bulb-like growth where it attaches to its host root.

Striga Control

Control of *Striga* in sorghum fields is difficult. No single control method is completely satisfactory in all cases. It is important to stress that *Striga* control is a long-term, continuing project and that a lapse in any segment allows the *Striga* to produce a seed crop, reinfect the field, and negate all previous efforts. Where possible, crop rotation with nongramineous crops over a period of several years greatly reduces the amount of *Striga* seed in the soil and facilitates further control. Hand weeding is practical in small areas where labor is available. Several weedings should be carried out once emergence begins, as *Striga* plants emerge over a considerable period of time. It is imperative that all *Striga* plants be pulled before seed is set; otherwise, the soil becomes reinfected with seed which will result in attack to future crops.

Mechanical cultivation in large areas reduces, but does not eliminate, *Striga* populations. The use of some of the new herbicides, judiciously applied, offers promise of excellent *Striga* control.

The development and introduction of *Striga*-resistant sorghum varieties also offer promise for *Striga* control in the future. These sorghum varieties offer two kinds of resistance. With one type, roots of the sorghum variety do not produce the stimulant for *Striga* seed germination. With the other type of resistance, the sorghum variety suppresses or prevents emergence of the *Striga* plants, preventing seed production. Neither of these two types of resistance is complete in every case, but they go far in reducing levels of *Striga* seed population. However, there is now evidence that races of *Striga hermonthica* in West Africa may attack sorghum in some areas; millet in other areas; maize in still other areas; and all of these crops in still other areas. How resistant sorghum varieties do against some of these races is still not known.

One of the most practical means of reducing *Striga* injury to sorghum is to use high plant densities with high nitrogen fertilization. The exact mechanism for this type of control is not presently known, but *Striga* emergence is considerably reduced where the sorghum canopy is dense enough to shade the soil.

Insecticide Injury

Some sorghum varieties are susceptible to injury by insecticides. Methyl parathion, toxaphene, and some of the organic phosphates can severely injure susceptible sorghum varieties. Injury may occur when plants of susceptible varieties are sprayed for control of shoot fly or stem borer. Injury may also occur from drift with ultra-low-volume application of insecticides to groundnuts or other crops. Insecticide injury from drift may appear on susceptible sorghum plants up to 1 kilometer from treated fields.

Insecticide injury appears as irregular, circular or elliptical, water-soaked spots on any part of the leaves within 24 hours of the time the chemical is applied. These spots dry out and turn reddish to blackish-purple on the margins within 72 hours (fig. 49). When a large quantity of insecticide is applied to a susceptible variety, large portions of the leaves may be destroyed. Sorghum plants outgrow the injury and new growth is not affected. However, repeated applications of insecticides may severely stunt or kill plants of susceptible varieties. Insecticide injury often may be identified by noting that, where leaves of one plant cross those of another, the lower or protected leaf will show an unspotted band the width of the leaf above.

Varieties of sorghum are available that possess high levels of resistance to insecticides. If sorghum is to be planted near groundnuts or other fields where ultra-low-volume applications of insecticides are made, varieties that are known to be resistant to insecticides should be used.

Figure 49. Insecticide injury on a susceptible variety of sorghum.

Figure 50. Interveinal necrosis on sorghum leaves.

Interveinal Necrosis

Some sorghum varieties show a conspicuous striped condition known as interveinal necrosis. This is a non-parasitic condition that appears on sorghum plants as long, wavy lesions almost the length of the leaf (fig. 50). Lesions are from 1 to 3 mm wide and are delimited by leaf veins. Lesion color ranges from red or purple-black to tan and brown. Lesions normally appear on sorghum plants just before they flower; sometimes they appear earlier. When interveinal necrosis appears on a variety, all or most of the plants in a field of that variety show symptoms. Sometimes interveinal necrosis appears on plants on one part of a sorghum field and not on plants in other parts of the field.

The exact nature of interveinal necrosis is not known; however, it is probably a physiological disorder. Since interveinal necrosis is restricted only to certain sorghum varieties, control may be easily obtained by planting only seed from varieties or plants that do not show this condition.

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